SHORT REPORTS

Toxic myocarditis in paracetamol poisoning

We describe the case of a young girl who died in cardiac failure 80 hours after a paracetamol overdose. Necropsy showed congested lungs, a dilated left ventricle, and myocardial muscle necrosis.

Case report

A 15 year old girl was admitted 55 hours after taking an unspecified quantity of paracetamol. She was fully conscious and oriented. Her pulse rate was 88 beats/min regular and blood pressure 110/70 mm Hg. The liver edge was palpable and tender. Plasma paracetamol concentration was 41 mg/l, serum sodium concentration 138 mmol/l, serum potassium concentration 4·2 mmol/l, plasma urea concentration 3·0 mmol/l, plasma bicarbonate concentration 10·0 mmol/l, prothrombin time 65 seconds, serum bilirubin concentration 56 μmol/l, serum alanine aminotransferase activity 790 IU/l, and serum aspartate aminotransferase activity 1075 IU/l.

Over the next 12 hours her plasma bicarbonate concentration improved to 16·6 mmol/l but serum potassium concentration decreased to 3·9 mmol/l. Serum creatinine concentration was 115 μmol/l at 15 hours, and urinary output exceeded 2 l in the first 21 hours. She received an intravenous infusion of dextrose, oral neomycin, vitamin K, cimetidine, and lactulose. Her blood pressure remained stable at 100/70 mm Hg, but persistent sinus tachycardia was recorded up to 21 hours after admission, when there was a sudden onset of cardiac arrhythmia and hypotension. Various atrial and ventricular arrhythmias were recorded. Electrocardiography showed gross ST segment depression and T wave inversion in leads 2, 3, aVF, V1-4. She became unconscious, with dilated pupils and absent brain stem reflexes. Spontaneous breathing ceased, and intermittent positive pressure ventilation was started. She went into ventricular tachycardia, followed by cardiac asystole, 25 hours after admission.

Necropsy showed a dilated left ventricle, pale myocardium (particularly subendocardially), which was soft in consistency; patent coronary arteries; and normal valves. Myocardial histology showed focal infiltrates of neutrophils and occasional mast cells among necrotic myocardial muscle fibres. Both lungs were diffusely oedematous. The liver was soft and showed punctate areas of centrilobular necrosis. Liver histology showed almost complete necrosis of hepatocytes, most pronounced in the centrilobular zones, with vacuolation of necrotic cells around the lobules. Pigment laden macrophages were present, but no inflammatory infiltration was found. The kidneys were slightly swollen with pale cortices. The brain was diffusely oedematous, but there was no coning of the brain stem and no evidence of necrotising myopathy.

Comment

The combination of hypotension, cardiac dilatation, and pulmonary congestion, in the absence of brain stem compression, made acute cardiac failure the immediate cause of death in this patient. Left ventricular dilatation is not a feature of fulminant hepatic failure, and this finding thus suggests toxic myocarditis. The absence of focal necrotising myopathy, a normal serum potassium concentration, and the improved plasma bicarbonate concentration[1] also indicate that cardiac failure was not related to the general metabolic disturbance.

Two previous reports of myocardial necrosis in paracetamol poisoning were questioned because other factors might have contributed to the outcome.[2,3] Dixon evaluated 20 unselected fatal cases of paracetamol poisoning confirmed by toxicology and claimed to have found no obvious cardiotoxicity, yet more than a quarter of these patients died from unexplained cardiac arrest.[4] He did not define his criteria for cardiotoxicity, and his conclusion must also be questioned. More importantly, he found no correlation between hepatic necrosis and death from paracetamol poisoning. Indeed, five of the patients he studied showed no hepatic necrosis at necropsy. Three others died from cardiac arrest, and a further three died from cerebral anoxia, which was itself a result of earlier cardiac arrest or inhalation of vomit. Only three patients died in unequivocal hepatic failure. He concluded that hepatic failure was not necessarily the cause of death in paracetamol poisoning and suggested the "acute toxicity" effect.

The number of patients who died from cardiac arrest in this small sample is large enough to suggest an alternative mechanism. The earliest histological change in the cardiac muscle after coronary artery occlusion may be detected five hours later,[5] or at 30 minutes with a special stain. Consequently, the absence of morphological evidence of cardiotoxicity in the six patients dying from cardiac arrest in Dixon's series does not rule out cardiotoxicity, assuming that standard histological techniques were used. In our case the finding of cardiac dilatation at necropsy and polymorph infiltration of the myocardium provides a helpful lead in this direction.

We thank Professor R A Risdon, professor of pathology at the Institute of Child Health, London University, for providing us with a copy of his postmortem report.

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3 Pimstone BL, Uys CJ. Liver necrosis and myocardiopathy following paracetamol overdosage. *S Afr Med J* 1968;42(II):259-62.
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5 Olsen EGJ. Myocardial infarction. In: Silverman MD, ed. *Cardiovascular pathology*. Vol 1. Edinburgh: Churchill Livingstone, 1983:406-19.

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Hyperthyroidism and de Clérambault's syndrome in a young woman

The features of de Clérambault's syndrome,[1] or erotomania, are a delusional belief of being loved by another person, usually of a higher status, who has been the first to express love and make advances. The onset is sudden, and the object of the delusion of love remains unchanged. In addition, the patient gives an explanation for the paradoxical behaviour of the loved one.

This chronic syndrome may be a primary disorder or secondary to other illnesses, such as schizophrenia, mania, alcoholism, or epilepsy. Ellis and Mellsop, reviewing reports of the syndrome, noted that the primary disorder was rare and that most of the cases were of secondary erotomania.[2] There are no previous reports of erotomania in patients with hyperthyroidism.

Case report

The patient, a 36 year old, single woman, worked as a clerk in a government office. She complained that her colleagues had laughed when she confided in them that the director was in love with her. Feeling hurt, she had started to cry and had accused them of being jealous.

She had a five year history of hyperthyroid Graves' disease but was non-compliant with treatment, and her attendance at the follow up clinic was irregular.

On admission she was restless and irritable. She had lost 5 kg in four months, although her appetite was good. There was a fine tremor of the hands, and her pulse was 96 beats/min. The thyroid gland was diffusely enlarged and soft, and a bruit was heard.

Thyroid function tests indicated thyrotoxicosis, thyroxine concentration being 156 nmol/l (normal range 55-145), thyroid stimulating hormone <0·5 mU/l (normal range 0·5-4·0), thyroxine binding globulin 186 nmol/l (normal range 168-324), and the ratio of thyroxine to thyroxine binding globulin 8·4 (normal range 2·8-4·8).

During the interview she mentioned that about two months previously she had noticed that whenever the director entered the office he would look at her. She also said that every morning he followed the bus that she took to work in his car. They had never spoken to each other, but she thought that this was "because he was a married man and it could be very embarrassing." She had sent 14 letters to him, and in one letter she wrote: "You loved me first and I have responded." When he did not reply she wrote again saying, "I hate you, but I know you are in love with me."

She came from a family of four children and was the only daughter. She had no steady boyfriend and worried that she might not get married. Except for the hyperthyroidism there was no important medical or psychiatric history.

The director said that he had not known her previously; there were about 300 workers in the department and he could not remember every face or name.

She was treated three times daily with carbimazole 10 mg, propranolol 20 mg, and chlorpromazine 50 mg. She responded well and was euthyroid after four weeks. The delusion of love persisted until the fifth week after admission, when she began to realise that "the whole thing is quite absurd and very embarrassing to him and me." After she was discharged she agreed to be transferred to another office "to avoid meeting him or my colleagues."

She was followed up regularly for almost two years and showed no signs of relapse. She stopped taking antithyroid drugs several times and suffered brief relapses of hyperthyroidism, but without any psychosis.

Comment

Hyperthyroidism commonly presents with mental changes such as irritability and anxiety, but psychotic symptoms like hallucination and delusion are rare. The review by Ellis and Mellsop shows that de Clérambault's syndrome is also rare.

Our case satisfied all the criteria of the syndrome, but it did not run a chronic course. The prognosis of the primary disorder is generally poor,[2 3] but in this case of secondary erotomania the outcome was good.

1 De Clérambault GG. Les psychoses passionelles. In: *Oeuvres psychiatriques*. Paris: Presses Universitaires de France, 1942:315-22.
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Asphyxiation by a child's dummy

All dummies sold in the United Kingdom conform to British Standard 5239. We report a case in which a dummy caused asphyxia in an 8 month old boy.

Case report

An 8 month old boy, who had used a dummy for seven months, presented with cyanosis but was still making some respiratory effort. The flange of a dummy was wedged behind the posterior tonsillar pillar, and there was a small amount of intraoral blood. The handle (ring) of the dummy was missing, having broken off at the hinge adjacent to the flange. Intraoral digital pressure on one side of the flange caused it to pivot, its edge was gripped with a towel clip, and the dummy was extracted. Suction removed the oropharyngeal blood, and he cried and became pink. He was given oxygen by facemask. His chest was clear on auscultation, and an *x* ray film four hours after admission showed no swelling of the soft tissue in the upper airway. Observation for 24 hours was uneventful.

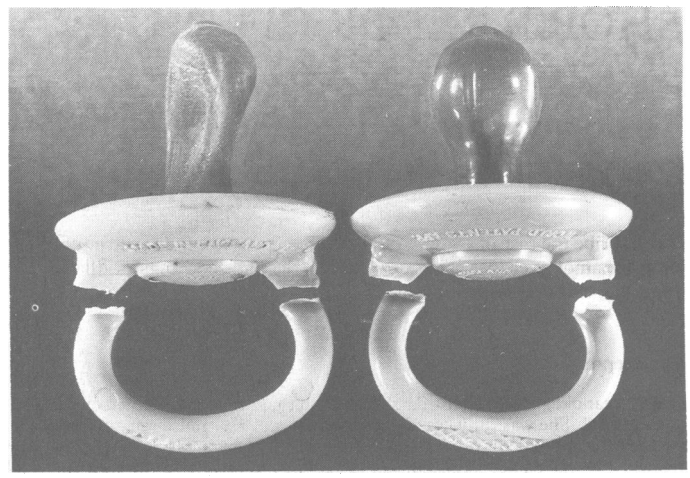
Before presentation he had often had part of his dummy's flange in his mouth, behind either the upper lip or the superior alveolar ridge. Occasionally only the ring would be visible out of his mouth. This time cyanosis was noted when odd gurgling sounds alerted his parents. The ring, still visible between his lips, moved gradually further into his mouth as if he were swallowing it. His father attempted to remove the dummy by grasping the ring and pulling. The ring detached at the flange, which filled out most of his mouth so his father could not extract it. The ambulance crew, attempting digital removal by inverting the child and using the Heimlich manoeuvre, failed to dislodge it. He was given oxygen, and oral suction was performed.

Although the various attempts to remove the dummy may have forced it into the oropharynx, rather than his swallowing on the teat having pulled it there, the relative ease with which it was removed indicates that the oropharynx was big enough for the flange.

THE DUMMY

The dummy was a blend of polypropene-polyisobutene, tough and strong under normal conditions; its fractured surface showed some evidence of strain whitening.

Twelve identical dummies, bought and tested according to the mechanical properties section (7) of British Standard 5239, conformed to the British Standard. Another 12 dummies were studied for the effects of (*a*) deforming at high strain rates and (*b*) fatiguing before (*a*). For British Standard 5239 the ring must withstand a load of 60 N applied over five seconds perpendicular to the main axis and maintained for 10 seconds, a grip separation rate of 0·5 mm/s. Under these conditions, the load causing fracture was 260 N. When the grip separation rate was increased to 5 mm/s the load causing fracture was only 60 N, the fractured surface being almost identical with that of the dummy removed from the child (figure). Fatiguing the dummy (bending the ring through 180° 1000 times) and then stressing it at the higher strain rate increased the load causing the fracture but reduced the ductility.



Fractured surfaces of dummy that caused asphyxiation with a ring broken off during testing (left) and of one subjected to high strain rate (grip separation rate 5 mm/s, load 60 N) (right).

Comment

The ring probably fractured because of the rapid deformation resulting from the father's tugging at the dummy, trying to remove it from the throat of his choking child.

British Standard 5239 considers only a low strain rate; we recommend that dummy rings should withstand a load equal to or greater than 120 N, with a grip separation rate of 5 mm/s. We suggest also that further consideration be given to the size and shape of flanges because of the ease with which the whole of the standard flange entered the baby's mouth and oropharynx. No radiographic data exist on the normal measurements of the oropharynx, except for the length of the hard palate and depth of the posterior pharyngeal wall,[1] primarily because of the variation in amount of soft tissue and its elasticity.

We thank the British Standards Institution for its cooperation.

1 Keats TE. *Atlas of roentgenographic measurement*. Chicago: Year Book Medical Publishers, 1985.

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Testicular relapse after chemotherapy for malignant teratoma

Extragonadal presentation of malignant teratoma is well recognised.[1] The treatment is usually cytotoxic chemotherapy, which is highly successful. We report here on a patient who had extragonadal metastatic teratoma and whose testis subsequently relapsed.

Case report

A 23 year old man presented with severe abdominal pain and left supraclavicular lymphadenopathy. No testicular abnormality was noted. Laparotomy showed a large retroperitoneal mass displacing the stomach and duodenum. Biopsy of the tumour showed undifferentiated malignant teratoma. Serum β human chorionic gonadotrophin and α fetoprotein concentrations were increased at 2785 IU/l and 25 IU/l, respectively. Computed tomography showed no evidence of tumour spread to the lung or liver. He received five courses of a three week chemotherapy